Endocrine Emergencies

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KEYWORDS

• Endocrine emergencies • Thyrotoxicosis • Myxedema coma • Addisonian crisis • Ketoacidosis • Pituitary • Hypercalcemia • Hypocalcemia

KEY POINTS

- Endocrine emergencies span a range of conditions involving glands including the thyroid, parathyroid, pituitary, pancreas, and adrenal glands, characterized by either an excess or deficiency of hormone levels.
- Clinical presentation may be nonspecific, which may delay diagnosis. However, it is crucial to begin treatment without delay before a diagnosis is made.
- Management involves a team approach. Therefore, enhanced awareness among health care professionals for swift intervention in these critical scenarios can decrease mortality rates.

THYROID EMERGENCIES: THYROID STORM AND MYEDEMA COMA

Thyroid Storm or Acute Thyrotoxicosis

Definitions

Hyperthyroidism. Elevated levels of thyroid hormones in the blood may occur as the result of increased synthesis and release of thyroid hormones from the thyroid gland.[1]

Thyrotoxicosis. Clinical manifestations and symptoms resulting from increased thyroid hormones in the blood. Severity may range from overt thyrotoxicosis to more severe thyroid storm.[2]

Thyroid storm. Acute, life-threatening condition characterized by an exaggerated manifestation of thyrotoxic state resulting in end-organ damage or decompensation.[2]

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Epidemiology

In the United States, the incidence of thyroid storm is 0.57 to 0.76 per 100,000 persons per year, with a higher incidence in hospitalized patients (4.8–5.6/100,000). The average age is 42 to 43 years, and the male:female ratio is 1:3.[3]

Causes and triggers

There are many factors including infection, trauma, and abrupt discontinuation of thioamide (methimazole, carbimazole, propylthiouracil) therapy that could potentially trigger the development of thyroid storm. These are summarized in **Table 1**.[4,5] Underlying Graves' disease is noteworthy as it may increase the risk of acute thyroid storm; however, 24% to 43% of the cases of thyroid storm have no identifiable trigger.

Presentation

The clinical presentation during thyroid storm is often variable and patients may present with nonspecific findings. **Fig. 1** shows features commonly present at the time of diagnosis. Most findings are due to the amplified effects of the thyroid hormone causing increased oxygen consumption and increased activity of sympathetic nervous system.

Evaluation and diagnosis

The diagnostic laboratories and imaging tests are summarized in **Table 2**. The diagnosis of thyroid storm must be made based on clinical findings. Moreover, laboratory tests to determine thyroid function may not be significantly different from other thyrotoxic states and therefore inconclusive when trying to diagnose thyroid storm. Fever (>39 C or 102°F) is the most common sign, and thyroid storm should be highly considered in a patient presenting with fever and known thyroid disease.[5] Diagnostic criteria such as the Burch–Wartofsky point scale may assist in diagnosis; however, it should not be used in isolation as a high score lacks specificity to thyroid storm and can be seen in other conditions such as sepsis.[6]

Management

Thyroid storm is a life-threatening condition with high mortality if left untreated; therefore, effective treatment must be initiated prior to test results. Initial management involves admission to the intensive care unit (ICU), decreasing body temperature using antipyretics, and volume resuscitation using isotonic intravenous (IV) fluids to account for fluid loss secondary to vomiting or diarrhea.[7] Medical therapy includes beta-blockers, antithyroid hormone therapy, inorganic iodide, and corticosteroid

Table 1
Identified triggers of thyroid storm[4]

Thyroid disease	Graves' disease, toxic multinodular goiter
Infection:	Any bacterial or viral infection, postviral thyroiditis, and suppurative thyroiditis
Pregnancy related	Pregnancy, labor, postpartum thyroiditis, struma ovarii, and molar pregnancy
Stress:	Trauma, burns, MI, PE, stroke, DKA, and intense exercise
Malignancy:	Metastatic or benign thyroid cancer resulting in goiter
Iatrogenic	Discontinuation of antithyroid medications, overdose of thyroid hormone, amiodarone, thyroid surgery
Toxins:	Certain drugs such as salicylates, organophosphates

Abbreviations: DKA, diabetes ketoacidosis; MI, myocardial infarction; PE, pulmonary embolism.

Fig. 1. Common clinical presentation of thyroid storm.[2,4,5]

therapy. Since infection is one of the most common precipitating factors, the American Thyroid Association (ATA) recommends starting an empiric broad-spectrum antibiotic. Medical therapy is aimed at the following mechanisms[8]:

1. To control an increased adrenergic tone, beta-blockers with a short half-life, for example, propranolol and esmolol are preferred. However, the use of beta-blockers should be avoided in acute decompensated heart failure with systolic dysfunction.
2. To reduce thyroid hormone synthesis and release, the use of thioamides should be considered. Propylthiouracil (PTU) is preferred over methimazole due to its impact on T4 to T3 conversion. Inorganic iodine solution should only be administered after thioamide initiation.

Table 2
Common laboratories and imaging to obtain when suspecting thyroid storm[5,6]

Laboratory/Imaging	Reasoning
CBC, inflammatory markers, urinalysis, chest X-ray (CXR)	To rule out infection, anemia, and assess for heart failure
Thyroid-stimulating hormone (TSH), T3, and free T4	To evaluate thyroid function. May be inconclusive
Complete metabolic profile, CK	To check for renal and liver function. Electrolyte disturbances may trigger thyroid storm
EKG	Arrhythmias such as atrial fibrillation, common in 10%–30% cases of thyroid storm
Urine drug screen	Certain toxins may trigger thyroid storm
Beta-human chorionic gonadotropin	Pregnancy and certain conditions during pregnancy may precipitate disease
Computed tomography (CT) head	To rule out neurologic causes (such as pituitary adenoma)

Abbrevitions: CBC, complete blood count; CK, creatinine kinase; EKG, electrocardiogram.

3. To block peripheral conversion of T4 to T3, one should use corticosteroids. PTU and beta-blockers also help with blocking the peripheral conversion of T4 to T3.
4. Reduce enterohepatic recycling of thyroid hormone by using bile acid sequestrants (eg, cholestyramine).

Effective management can be summarized with the mnemonic "B.A.S.I.C.":

B: B-adrenergic blockers, broad-spectrum antibiotics
A: Antithyroid hormone therapy
S: Sequestrants (bile acid sequestrants)
I: Inorganic iodide, ICU admission, IV fluids
C: Corticosteroid therapy

Myxedema Coma

Introduction

Myxedema coma is defined as decompensated hypothyroidism leading to multiorgan failure.[9] The exact incidence is unknown; however, studies show it ranges from 0.22 to 1.08 cases per million patients per year. Women aged above 60 years and those with a history of hypothyroidism are most affected.[10] "Myxedema coma" is often considered as a misnomer as the prime manifestation of the disease is deterioration of mental status, and most patients neither present with nonpitting edema (myxedema) nor coma.

Among these causes, failure to continue thyroid replacement therapy and infections are the most common risk factors that lead to the development of myxedema coma.[11,12] **Table 3** lists common precipitating factors of myxedema coma.

Clinical presentation

The clinical presentation and findings can be accounted for by the loss of thermogenesis, reduced metabolism, depression of the central respiratory system, and decreased gluconeogenesis.[13] The neurologic change may present as lethargy and obtundation or an activated state known as "myxedema madness."[14] **Fig. 2** summarizes typical features at presentation while **Table 3** indicates common precipitating factors.

Diagnosis

If a patient with known hypothyroidism presents with depressed mental status and hypothermia in the setting of a precipitating event, myxedema coma should be highly considered.[15] Diagnostic criteria are available but have not been widely assessed on different patient populations.[16] Recommended laboratory work and imaging include

Table 3
Common precipitating factors of myxedema coma[11,12]

Medications	Discontinuation or inability to start thyroid medications in a patient with known hypothyroidism or the use of sedatives, narcotics, and anesthetics
Infections	Most commonly pneumonia or urosepsis
Cardiovascular	Congestive heart failure
Obstetric	Labor
Neurologic	Cerebrovascular events
Environment	Low temperature
Trauma	Fractures, injury

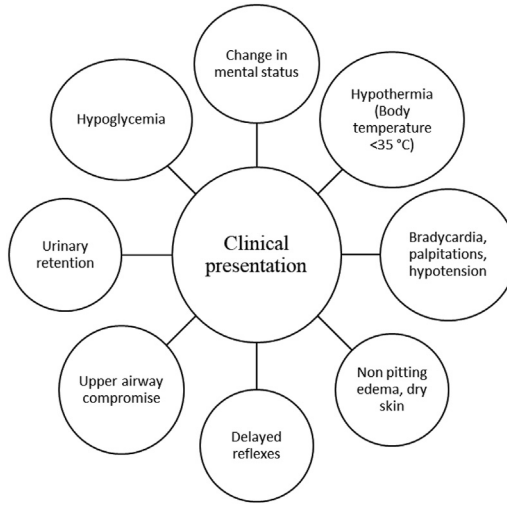

Fig. 2. Clinical presentation of myxedema coma.[13,14]

- CBC, inflammatory markers, urinalysis: to evaluate for infectious processes
- Arterial blood gases: to diagnose respiratory acidosis, hypercapnia, and hypoxia
- TSH, T4, T3: to evaluate thyroid function (most patients have elevated TSH and low T4)
- CK: to rule out rhabdomyolysis resulting from urinary retention
- Complete metabolic profile: electrolyte abnormalities specifically hyponatremia
- EKG: evaluate for arrhythmias, heart block, and torsades de point
- Echocardiogram and CXR: for pleural effusion or cardiomegaly
- MRI pituitary: to look for causes of secondary hypothyroidism (pituitary macroadenoma, empty sella)

Management

It is essential to initiate lifesaving treatment prior to waiting for laboratory results as that may delay the treatment course. An outline of general management of myxedema coma is shown in **Fig. 3**. Management includes admitting the patient to the ICU for continued monitoring and beginning prompt thyroid replacement therapy. A combination of parenteral levothyroxine and liothyronine is recommended.[17,18] Typically, one can start with a load of 200 to 400 mg of levothyroxine IV, followed by 50 to 100 mg IV daily. Liothyronine is administered concomitantly 5 to 20 mcg as loading dose

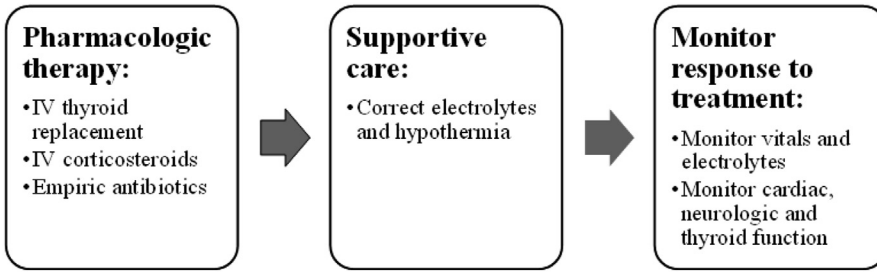

Fig. 3. Outline of the management of myxedema coma.[17–19]

followed by 2.5 to 10 mcg IV every 8 hours. The ATA recommends that after the initial loading doses, up titration in response to symptoms and switch to oral as soon as patient is able to tolerate oral.[18]

In addition to thyroid replacement, electrolyte abnormalities such as hyponatremia should be corrected with IV normal saline infusion. Passive rewarming methods including blankets should be to raise body temperature and prevent life-threatening hypothermia. Active rewarming methods using heated blankets are not recommended and may result in worsening hypotension due to peripheral dilation. Infections such as pneumonia and urosepsis are a precipitating factor for myxedema coma; therefore, empiric antibiotics should be started early. Moreover, IV corticosteroid therapy (hydrocortisone 100 mg then 50 mg q6hours) should be initiated until coexisting adrenal insufficiency is ruled out. Cardiac function, urinary output, neurologic function, vitals, and thyroid function should be closely monitored. Eventually, patients can be transitioned to oral thyroid replacement therapy based on clinical improvement.[19]

PARATHYROID EMERGENCIES: HYPERCALCEMIC CRISIS AND HYPOCALCEMIC CRISIS

Hypercalcemic Crisis

Epidemiology and pathophysiology

Hypercalcemic crisis is an uncommon condition in which patients develop severe hypercalcemia with signs and symptoms of multiorgan dysfunction. Primary hyperparathyroidism, is one of the most common causes of hypercalcemic crisis.[20]

Presentation

Common signs and symptoms of hypercalcemia crisis may be general (generalized weakness and malaise) musculoskeletal (bone pain, fractures, and muscle weakness), nephrogenic (anuria, oliguria, nocturia, nephrolithiasis, and acute renal failure), or gastrointestinal (abdominal pain, constipation, and vomiting).[21] Patients may also present with complications of hypercalcemia including pancreatitis, nephrolithiasis, and cardiac arrhythmias.

Diagnosis and workup

Diagnostic workup for hypercalcemia can be extensive. **Table 4** lists common laboratory tests and imaging procedures that may be done to determine the etiology of hypercalcemic crisis and to monitor the resulting end-organ dysfunction.

Hypoalbuminemia may alter calcium levels in blood; therefore, it is important to calculate corrected blood calcium using the following formula.

$$\textit{Corrected calcium} = \textit{serum calcium} + 0.8 \times (4 - \textit{serum albumin})$$.[22]

An exact cutoff to diagnose hypercalcemic crisis has not been decided; however, the calculated albumin-corrected blood calcium level in hypercalcemic crisis is usually greater than 14 mg/dL. [23]

Management

The mortality rate due to parathyroid crisis was previously reported to be as high as 66%; however, it has decreased over time due to early recognition and improved treatment.[24] If calcium level persistently remains critically elevated, the organs most affected and prone to decompensation are the brain and kidneys, potentially resulting in irreversible coma or renal failure.[25]

Therefore, early recognition of the disease and initiation of treatment is important. Medications such as digoxin and hydrochlorothiazide should be avoided as they increase mortality.[20] Preoperative supportive measures that have demonstrated efficacy in improving the clinical condition of patients include[25,26]

Table 4
Common workup in suspected hypercalcemic crisis[6]

Laboratory Tests	Rationale
Calcium, ionized calcium, and albumin	To confirm hypercalcemia and calculate corrected calcium
Parathyroid hormone (PTH)	To differentiate between PTH-mediated and non-PTH-mediated etiologies
25-hydroxyvitamin D	To evaluate the impact of vitamin D deficiency-mediated secondary hyperparathyroidism on PTH level
Complete metabolic profile	Elevated BUN and creatinine are often seen in hypercalcemia. To evaluate liver function, kidney function, and to plan electrolyte repletion
Urine studies (including urine protein) PTHrP, 1,25-dihydroxyvitamin D and SPEP/UPEP	To evaluate non-PTH-mediated hypercalcemia etiologies
Imaging	
EKG	To evaluate for arrhythmias
Parathyroid sestamibi scan and neck ultrasound	Performed for localization of enlarged parathyroid glands, once primary hyperparathyroidism has been confirmed biochemically

Abbreviations: PTHrP, parathyroid hormone related peptide; SPEP, serum protein electrophoresis; UPEP, urine protein electrophoresis.

1. Rehydration: Aggressive IV rehydration with isotonic saline to counteract hypercalcemia-driven osmotic diuresis and argenine vassopressin (AVP) resistance.
2. Calcium lowering therapy: IV calcitonin and IV infusion of bisphosphonates such as pamidronate and zoledronic acid. Denosumab is the preferred agent among patients with severe acute kidney injury (AKI).
3. Loop diuretics: to promote calciuresis, only if patient appears to have fluid overload.

Hypercalcemic crisis secondary to hyperparathyroidism is considered a surgical emergency and surgical parathyroidectomy within 24 to 72 hours remains the definitive treatment option.[21] Therefore, ectopic release of parathyroid hormone must be ruled out, and the diagnosis must be established early to plan for necessary surgical treatment.

Hypocalcemic Crisis

Epidemiology and etiology

Hypoparathyroidism is an uncommon condition, with an estimated incidence of 37 per 100,000 person-years in the United States and can result in potentially life-threatening hypocalcemia.[27] Anterior neck surgery accounts for about 75% of cases of hypoparathyroidism, but it can also be due to autoimmune conditions, radiation, and metastatic disease.[28]

Clinical Presentation

Table 5 lists signs and symptoms of acute hypocalcemia.[29]

Diagnosis

Laboratory testing aims to assess parathyroid function and exclude other electrolyte abnormalities (hypomagnesemia) or disorders that might manifest with comparable

Table 5
Common clinical presentation of acute hypocalcemia

Neuromuscular excitability	Chvostek's sign, Trousseau's sign, paresthesias, tetany, seizures (focal, generalized), muscle cramps and weakness, dystonic spasms, laryngospasm, and bronchospasm
Mental status	Confusion, disorientation, psychosis, personality changes, irritability, impaired intellectual ability, and parkinsonism
Cardiac	Palpitations, signs of congestive heart failure (dyspnea and edema)
Smooth muscle involvement	Dysphagia, abdominal pain, biliary colic, dyspnea, and wheezing

clinical signs. Initial laboratory analysis involves assessing serum levels of calcium, phosphate, magnesium, intact PTH, 25-hydroxyvitamin D, and 1,25-dihydroxyvitamin D alongside measuring albumin level to calculate corrected serum calcium.[29] **Table 6** lists recommended investigations to aid in the diagnosis of hypocalcemia.

Treatment

Acute hypocalcemia is considered an emergency, requiring immediate attention. If not addressed promptly, acute hypocalcemia has the potential to rapidly advance, resulting in diverse neurologic and cardiac complications that can ultimately lead to death. Complications include seizures, status epilepticus, coma, or life-threatening cardiac arrhythmias.[30]

The typical threshold for initiating acute management is a calcium level of 1.9 mmol/L (7.5 mg/dL)[30]; however, the decision to administer acute therapy is also guided by symptoms. Emergent treatment of hypocalcemia with IV calcium gluconate is preferred, as IV calcium chloride often causes local irritation. The infusion should be continued until the patient is asymptomatic or calcium level returns to normal. In patients with end-stage renal failure or those on dialysis, large-volume calcium infusions should be avoided. Potential hazards of IV calcium administration, although uncommon, include local thrombophlebitis, cardiotoxicity, hypotension, calcium taste, flushing.[31]

Prevention

Some protocols advocate administering calcium and calcitriol postoperatively after thyroid surgery, regardless of serum calcium levels, to reduce the risk of developing hypocalcemia. Additionally, perioperative PTH concentration, preoperative vitamin

Table 6
Common workup for suspected acute hypocalcemia crisis[29]

Laboratory Tests	**Rationale**
Serum total calcium, ionized calcium	Assess extent of hypocalcemia
Phosphate	Assess for hypophosphatemia
PTH	Assess for primary hypoparathyroidism
Magnesium	Hypomagnesemia may lead to impaired release of PTH
Vitamin D levels	Low vitamin D levels result in low calcium absorption
Electrocardiogram	Hypocalcemia leads to prolonged Q wave to T wave (QT) and arrhythmias
Neck ultrasound	Investigate structural abnormalities of parathyroid gland
Urine calcium	To distinguish between renal and nonrenal etiology

D levels, and early postoperative changes in calcium levels serve as useful biochemical predictors of postthyroidectomy hypocalcemia.[32]

ADRENAL EMERGENCIES: ADRENAL CRISIS AND PHEOCHROMOCYTOMA

Adrenal Crisis/Addisonian Crisis

Introduction

An adrenal crisis is a critical endocrine emergency that arises from the insufficient production of the adrenal hormone cortisol, a primary glucocorticoid. This life-threatening condition can stem from either primary adrenal insufficiency, characterized by the impaired function of the adrenal gland itself, or secondary adrenal insufficiency, where the regulation of adrenal cortisol production by the pituitary is compromised.

In adults, an adrenal crisis is characterized by a sudden decline in health marked by either absolute hypotension (systolic blood pressure <100 mm Hg) or relative hypotension (systolic blood pressure ≥20 mm Hg lower than usual). This condition is typically associated with symptoms that show marked improvement within 1 to 2 hours after the administration of parenteral glucocorticoids.[33] Every year, around 6% to 8% of individuals with adrenal insufficiency experience an episode of adrenal crisis. Literature indicates a prevalence of adrenal crisis ranging from 5.2 to 8.3 per 100 patient years.[34]

Causes and triggers

A number of factors have been implicated in precipitating Addisonian or adrenal crisis. **Table 7** lists common precipitating factors leading to adrenal crisis.[33]

Clinical presentation

Patients may present with variable symptoms including abdominal pain, anorexia, nausea, vomiting, fatigue, postural dizziness, and impaired consciousness or coma.[35]

Diagnosis

Common laboratory findings include hyponatremia, hyperkalemia, hypercalcemia, hypoglycemia, neutropenia, eosinophilia, lymphocytosis, and normocytic anemia.[33]

If there is suspicion of an adrenal crisis, it is advisable to draw a blood sample promptly for serum cortisol and adrenocorticotropic hormone (ACTH); however, treatment should commence without waiting for assay results.[36] ACTH stimulation test, a diagnostic tool employed under stable conditions (involving the measurement of serum cortisol at baseline, 30 and 60 minutes after an IV injection of ACTH 250 μg), is of limited utility in emergency situations and could unnecessarily prolong the initiation of treatment.[37]

Treatment

Treatment should be initiated as soon as adrenal crisis is suspected as commencing lifesaving hydrocortisone treatment poses no adverse consequences. Treatment is aimed at the following[38]:

1. Glucocorticoid replacement: prompt administration of IV hydrocortisone 50 to 100 mg. This can be followed by hydrocortisone 50 mg IV every 6 hours or a continuous infusion.
2. Rapid rehydration: Rehydration involves a rapid IV infusion of 1000 mL of isotonic saline.
3. Treating the precipitating event: For example, treatment with antibiotics may be necessary in the setting of infection.

Following the successful management of an adrenal crisis, the hydrocortisone doses should be gradually tapered. An assessment for potential preventable triggers should be conducted, and the patient should be educated about preventive strategies.[36]

Table 7
Common triggers precipitating adrenal crisis[33]

Primary adrenal insufficiency	Stress induced	Infections, Trauma, surgery, and psychological stress
	Ischemia	Due to major blood loss
	Hemorrhage	Waterhouse–Friderichsen disease (secondary to *Neisseria meningitidis* infection), peripartum, disseminated intravascular coagulation (DIC), and anticoagulant use
	Iatrogenic	Nonadherence to glucocorticoid replacement therapy
Secondary adrenal insufficiency	Hypothalamic-pituitary related	Pituitary tumor, Sheehan's syndrome, and pituitary apoplexy
		Chronic glucocorticoid use

Pheochromocytoma Crisis

Introduction

Pheochromocytoma is a rare tumor that results in excess production of catecholamines and can lead to pheochromocytoma crisis, which can be classified as type A and type B. Type A crisis includes hemodynamic instability and evidence of end-organ dysfunction of least one-organ system. Type A (limited crisis) may progress to type B (extensive crisis and shock), which has a higher mortality rate, and is characterized by sustained hypotension and multiorgan dysfunction.[39]

Symptoms

Pheochromocytoma crisis typically presents with hypertensive emergency. Other conditions and complications associated with pheochromocytoma crisis include stroke, arrhythmias, acute coronary syndrome, pulmonary edema, respiratory failure, seizure, multiorgan failure, and death.[39]

Evaluation

Acute evaluation varies based on symptomatology, but typically includes a CBC, complete metabolic profile (CMP), TSH, toxicology, electrocardiogram, CXR, and abdominal CT.[39]

Treatment

Alpha blockade hypertensive emergency treatment is used to treat hypertension, and IV phentolamine is commonly recommended.[39] Other treatments vary based on the individual case and presentation and may include the need for ventilatory support.

PANCREATIC ENDOCRINE EMERGENCIES: DIABETIC KETOACIDOSIS

Part 1: Diabetic Ketoacidosis

Introduction

DKA is an acute complication of diabetes mellitus that carries a high-risk of mortality if not treated appropriately. It stands as the primary cause of mortality in individuals aged under 24 years with diabetes, primarily attributed to cerebral edema in many cases.[40] In approximately 25% to 40% of patients with type 1 diabetes, DKA may

manifest as the initial presentation.[41] Initially believed to be exclusive to individuals with type 1 diabetes, DKA has also been identified in patients with type 2 diabetes under specific conditions.

Pathophysiology

The primary triggers in most cases are the onset of new diabetes, infection, or noncompliance with prescribed treatment.[42] Additional precipitating factors include alcohol abuse, trauma, pulmonary embolism, and myocardial infarction.[43]

Signs and symptoms

Typical manifestations of DKA include[42]

1. General symptoms: reduced urine output, dry mouth, poor capillary refill, and decreased sweating
2. Respiratory: dyspnea, fruity smell in breath, and Kussmaul (labored, deep, and rapid breathing)
3. Gastrointestinal: vomiting, abdominal pain and tenderness, anorexia, and nausea
4. Neurologic: Altered mental status, general drowsiness, and focal neurologic deficits (may occur in severe cases as a sign of cerebral edema)

Diagnosis

The diagnostic criteria for DKA as per the guidelines of the American Diabetes Association is outlined in **Table 8**.

In addition to evaluating blood glucose levels, blood pH, serum bicarbonate, ketones, and calculated anion gap, further laboratory investigations are typically conducted to assess the underlying cause and potential complications. A sepsis workup is carried out assessing lactate levels, complete blood count (which may reveal leukocytosis), and complete metabolic profile (particularly assessing electrolyte imbalances such as hypokalemia and hyponatremia). Additionally, a comprehensive investigation includes urinalysis, blood culture, urine culture, and a chest radiograph to diagnose potential infections. Osmotic diuresis and natriuresis result in a general depletion of electrolytes. Consequently, an EKG is conducted to exclude arrhythmias that may arise as a secondary effect of electrolyte imbalances.

Management

Failure to treat DKA can lead to severe complications that may be fatal, including arrhythmias, cerebral edema, respiratory failure, and rhabdomyolysis. Hence, swift recognition of the disease and prompt initiation of treatment are imperative.

The therapeutic objectives for individuals experiencing hyperglycemic crises involve the following[41]:

Table 8
Diagnostic criteria for diabetic ketoacidosis as per American Diabetes Association guidelines[43,44]

	Mild	Moderate	Severe
Serum pH	7.25–7.30	7.00 to <7.24	<7.00
Blood glucose	>250	>250	>250
Serum bicarbonate (mEq/L)	15–18	10 to <15	<10
Urine and serum ketones	Positive	Positive	Positive
Anion gap	10	>12	>12
Mental status	Alert	Alert/drowsy	Stupor/coma

1. Administering IV fluids to improve tissue perfusion: Fig. 4 for a concise flowchart on IV rehydration.
2. Initiating insulin therapy to lower serum glucose: Start with a bolus of IV regular insulin 0.1 U/kg followed by 0.1 U/kg/h continuous infusion. The insulin infusion dose can be doubled if serum glucose does not fall by 50 to 70 mg/dL in the first hour of treatment. Adjust and reduce the insulin infusion to 0.05 to 0.1 U/kg/h once serum glucose reaches 300 mg/dL.
3. Repletion of potassium: Replete potassium before initiating insulin infusion if serum levels are below 3.3 mEq/L. For levels between 3.3 and 5.3 mEq, add 20 to 30 mEq of potassium to each liter of fluid administered. Monitor serum potassium every 2 hours if levels exceed 5.3.
4. Assessing the need for bicarbonate: Administer bicarbonate if serum pH is below 6.9; otherwise, no intervention is needed.
5. Promptly identifying and treating precipitating causes.

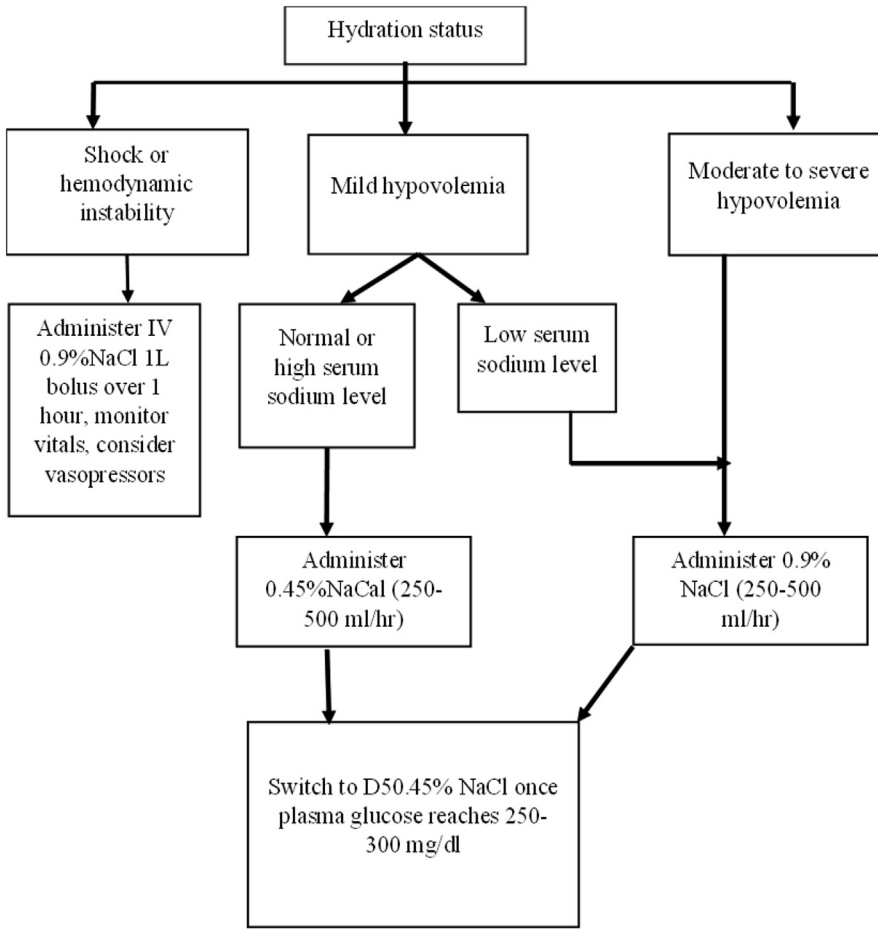
Hydration status
Shock or hemodynamic instability
Mild hypovolemia
Moderate to severe hypovolemia
Administer IV 0.9%NaCl 1L bolus over 1 hour, monitor vitals, consider vasopressors
Normal or high serum sodium level
Low serum sodium level
Administer 0.45%NaCal (250-500 ml/hr)
Administer 0.9% NaCl (250-500 ml/hr)
Switch to D50.45% NaCl once plasma glucose reaches 250-300 mg/dl

Fig. 4. Protocol for IV fluid administration for DKA.[41]

PITUITARY EMERGENCIES: PITUITARY APOPLEXY

Pituitary Apoplexy

Epidemiology

Pituitary apoplexy is a rare clinical syndrome where there is hemorrhage or infarct of the pituitary gland. It occurs in 2% to 12% of pituitary adenomas, with a higher prevalence among nonfunctioning macroadenomas.[45] If not identified and addressed promptly, pituitary apoplexy can pose a life-threatening risk with overall mortality rates between 1.6% and 1.9%.[46] Factors contributing to pituitary apoplexy may include surgical procedures resulting in reduced blood flow, stimulation applied to the pituitary gland, and the use of anticoagulant medications in patients.

Clinical presentation

Pituitary apoplexy commonly presents with a primary symptom of headache, often accompanied by visual field impairment or ocular palsies, with bitemporal hemianopsia being the most prevalent. They can present with cognitive changes and may manifest symptoms of meningeal irritation, such as photophobia, nausea, and vomiting.[47] Furthermore, multiple acute endocrine insufficiencies including deficiencies in anterior pituitary hormones like ACTH and TSH may occur simultaneously, leading to symptoms of acute adrenal and thyroid insufficiency.

Diagnosis

MRI is the preferred radiological investigation and has been proven effective in confirming the diagnosis of pituitary apoplexy.[47]

Treatment

In the management of pituitary apoplexy, providers should prioritize hemodynamic stability. Maintaining electrolyte balance, replacement of deficient hormone, and making referrals to endocrine and neurosurgery specialists are also crucial steps. Since corticotropic deficiency is prevalent in most patients, IV corticosteroids (IV hydrocortisone) should be promptly administered upon confirming the diagnosis.[48]

The decision to pursue conservative or surgical management should be made by a multidisciplinary team of experts. Surgery, often utilizing a trans-sphenoidal approach, has demonstrated the highest efficacy in addressing pituitary apoplexy.[49]

Prognosis

Surgical decompression typically leads to improvement in altered consciousness, and visual field, especially when they were normal before the acute episode. Ophthalmoplegia usually resolves, but the process may take several weeks. Endocrine function often remains mildly altered even after treatment; therefore, long-term follow-up, both endocrine assessment and imaging, is necessary.[45]

DISCLOSURE

The authors have nothing to disclose.

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